RESPONSES TO COMMENTS SUBMITTED BY THE NATURAL RESOURCES DEFENSE COUNCIL.

Comment 1: … We are quite concerned that 1,3-butadiene, an extremely important toxic air contaminant, is omitted completely from the draft prioritization document. 1,3-butadiene is important for at least two reasons: First, there is some evidence that developing animals are more susceptible to the adverse effects of butadiene compared to adults.[1] Second, the exposures and risks are very high – the EPA (prior to new attempts to down-grade the risk of butadiene based on a study of adult males) found that butadiene is one of the top five TACs (benzene, butadiene, chromium, PAHs, and formaldehyde) responsible for a cumulative total of 75% of the cancer risk in ambient air.[2] Estimated concentrations of benzene, formaldehyde, and 1,3-butadiene were greater than benchmark concentrations in over 90% of the census tracts in the United States.[3] In California, toxic air contaminants contributed a median cancer risk of 2.7 per 10,000 based on 1990 data, with an estimated 8600 excess cancer cases attributable to these hazards. rikingly, 70% of the total cancer risk was attributable to four pollutants: PAHs, 1,3-butadiene, formaldehyde, and benzene.[4] The South Coast Air Quality Management District estimated a cancer risk in that air basin of 1,400 per million people exposed. 70% of the risk was attributable to diesel exhaust, while an additional 20% was attributable to benzene, formaldehyde, and butadiene.[5] Therefore, NRDC strongly urges OEHHA to review the science relevant to children's disproportionate sensitivity to 1,3-butadiene and seriously consider this chemical for addition to Tier 1.

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[1] Morrissey RE, Schwetz BA, Hackett PL, et al. Overview of reproductive and developmental toxicity studies of 1,3-butadiene in rodents. Environ Health Perspect 86:79-84, 1990.

[2] Woodruff TJ, Caldwell J, Cogliano VJ, Axelrad DA. Estimating cancer risk from outdoor concentrations of hazardous air pollutants in 1990. Environ Res 82(3):194-206, 2000.

[3] Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106(5):245-251, 1998.

[4] Morello-Frosch RA, Woodruff TJ, Axelrad DA, Caldwell JC. Air toxics and health risks in California: the public health implications of outdoor concentrations. Risk Analysis 20(2):273-291, 2000.

Response: OEHHA is also concerned about the chronic health risks associated with exposure to 1,3-butadiene in ambient air. A butadiene toxicity summary draft has been prepared but was not included in the initial Prioritization public review draft. In prioritizing TACs OEHHA evaluated evidence for differential infant and child toxicity placing an emphasis on human data. In our view butadiene lacks sufficient data indicating differential infant and child toxicity that would replace any of the contaminants listed in Tiers one and two. The commenter cites Morrissey et al. (1990) as showing increased fetal sensitivity. In that study maternal toxicity was observed at the 200- and 1000-ppm butadiene levels, whereas developmental toxicity in the form of reduced male fetal weight was seen at the low dose level of 40 ppm. The weight reduction was only five percent relative to controls, albeit statistically significant ($p \leq 0.05$). This effect is considered relatively mild despite the high exposure level and does not provide strong evidence relative to the TACs selected in Tiers one and two. However, there are a number of features of butadiene toxicology that would support a concern for differential child toxicity. The target organs of BD particularly hematotoxicity and gonadal toxicity are of special concern. Also butadiene has demonstrated the ability to induce cancers in mice after only 13 weeks exposure in stop-exposure studies. OEHHA would expect to place butadiene in the next priority group.

Comment 2: Diesel Exhaust Should be in Tier 1.

NRDC recognizes that diesel exhaust contains many of the other toxic air contaminants in Tier 1 and Tier 2, most particularly the PAHs. There is evidence, however, that some of the health risks from diesel exhaust may be particular to that mixture. In particular, extensive recent research linking diesel exhaust to allergic and inflammatory responses in

[5] South Coast Air Quality Management District, *Multiple Air Toxics Exposure Study in the South Coast Air Basin* (MATES-II), March 2000.

the airways has generated serious concern that there may be a real.[6] In addition, according to the South Coast Air Quality Management District, diesel exhaust accounts for an estimated 70% of the cancer risk in ambient air.[5] These factors make it particularly important to protect children from health effects from this hazardous substance. We urge OEHHA to consider PAHs and diesel exhaust together as a group, as one of the top five priority toxic air contaminants in ambient air.

Response: OEHHA shares the commenter's concern over the potential for differential effects of diesel exhaust on infants and children, and concurs with the observation that diesel exhaust appears to have unique effects not readily attributable simply to PAHs or other TACs present in diesel exhaust. OEHHA considered the link between diesel exhaust and childhood asthma both in the toxicity summary included with the report, and in response to other commenters, has generally found it appropriate to rebut attempts to discount these findings. It might be procedurally difficult to adopt the commenter's suggestion that PAHs and diesel exhaust be combined for consideration under SB25, and in any case this may not be appropriate given the previous observation that diesel exhaust has unique effects not shared by PAHs in isolation. However, OEHHA is giving very serious consideration to the suggestion that diesel exhaust particulate matter be included as one of the five TACs listed in this prioritization.

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[6] Peterson B, Saxon A. Global increases in allergic respiratory disease: the possible role of diesel exhaust particles. Annals of Allergy, Asthma, and Immunology. 1996; 77:263-268; Wade JF, Newman LS. Diesel asthma: reactive airways disease following overexposure to locomotive exhaust. Journal of Occupational Medicine. 1993; 35:149-154; Takano H, Ichinose T, Miyabara Y, et al. Inhalation of diesel exhaust enhances allergen-related eosinophil recruitment and airway hyperresponsiveness in mice. Toxicology and Applied Pharmacology. 1998; 150:328-337. Ishizaki T, Koizomi K, Ikemori R, Ishiyama Y, Kushibiki E. Studies of prevalence of Japanese cedar pollinosis among residents in a densely cultivated area. Ann Allergy 58:265-270 (1987).

Comment 3: Benzene and Dioxin-Like Chemicals Should Remain in Tier 1.

NRDC strongly supports the placement of benzene and dioxin-like chemicals in Tier 1. The science supporting the exposures and health risks to children is relatively strong. There is no question (see butadiene discussion above) that benzene is among the most hazardous TACs. Previous studies have consistently placed this chemical among the top cancer risks in ambient air. In addition, the known link between benzene and leukemia makes this chemical of particular concern at a time when the incidence of childhood leukemia has been rising for two decades. A new study also supports the link between childhood acute lymphoblastic leukemia and solvent exposure.[7] One of the chemicals implicated in this study is benzene. We urge OEHHA to include this new study in the discussion of benzene's cancer risk to children.

Because benzene and 1,3-butadiene have numerous similarities, NRDC suggests that OEHHA group them together as a priority set of TACs. Both of these chemicals are primarily emitted from mobile sources, both behave similarly in the environment, and both are associated with hematological malignancies in humans. Although there are some areas of difference, we believe that it is scientifically and pragmatically justifiable to group these two chemicals for risk assessment and regulatory purposes.

Dioxin-like chemicals are known developmental toxicants. There is little question that they pose a particular risk to fetuses and infants. Although air is not the major direct exposure pathway to these chemicals, the dioxin-like chemicals that ultimately end up in breast milk and food usually originate as air emissions. Addressing dioxins as air pollutants is a sensible step consistent with the principle of decreasing pollution nearer the source.

[7] Freedman DM, Stewart P, Kleinerman RA, et al. Household solvent exposures and childhood acute lymphoblastic leukemia. Am J Public Health 91:564-567, 2001.

Response: OEHHA thanks the commenter for the support given to the concern expressed in the prioritization document over the possibility that benzene and dioxin-like compounds may have differential impacts on infants and children. OEHHA thanks the commenter for pointing out the recently published study by Freedman et al. (2001), and agrees that air is an important transport medium for dioxins even when the eventual human exposure occurs via other media.

As noted in comment 1, OEHHA has evaluated 1,3-butadiene and although this chemical has not been given the highest priority at this stage it remains a significant concern and may be given further consideration at subsequent stages of the process mandated by SB25.

OEHHA is not persuaded that the legislative mandate allows the inclusion of groups of TAC so that more than five agents could be included in the "Tier 1" priority group, except in the special case of classes of compound accorded a single listing such as the dioxin-like compounds, the PCBs, or (following the Federal HAP listing of polycyclic organic material) the PAHs and related compounds.